

## WEST Search History





DATE: Wednesday, July 11, 2007

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		<i>DB=PGPB,USPT; PLUR=YES; OP=ADJ</i>	
<input type="checkbox"/>	L8	("20020192163"  "6749834")[URPN]	0
<input type="checkbox"/>	L7	("20020192163"  "6749834")[URPN]	0
		<i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ</i>	
<input type="checkbox"/>	L6	L3 and (angina or infarct or myocardi\$ or restenois or heart or cardiac\$ or cardio\$ or arryth\$ or asthma or rectal)	41
<input type="checkbox"/>	L5	L4 and (angina or infarct or myocardi\$ or restenois or heart or cardiac\$ or cardio\$ or arryth\$ or asthma or rectal)	70
<input type="checkbox"/>	L4	L1 and (reductant or TCEP or \$ethylphosine)	82
<input type="checkbox"/>	L3	L2 and (reductant or TCEP or \$ethylphosine)	44
<input type="checkbox"/>	L2	L1 and (thiol or dithiol or dithiothreitol or DTT or "dihydrolipoic acid" or DHLA)	1712
<input type="checkbox"/>	L1	nitroglycerin or GTN or "glyceryl trinitrate"	9550

END OF SEARCH HISTORY

ANSWER 1 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN  
 ACCESSION NUMBER: 1999:524993 BIOSIS  
 DOCUMENT NUMBER: PREV199900524993  
 TITLE: Influence of redox compounds on nitrovasodilator-induced  
 relaxations of rat coronary arteries.  
 AUTHOR(S): Murphy, Michael E. [Reprint author]  
 CORPORATE SOURCE: Department of Pharmacology and Neuroscience, Albany Medical  
 College, 47 New Scotland Avenue, Albany, NY, 12208-3479,  
 USA  
 SOURCE: British Journal of Pharmacology, (Sept., 1999) Vol. 128,  
 No. 2, pp. 435-443. print.  
 CODEN: BJPCBM. ISSN: 0007-1188.  
 DOCUMENT TYPE: Article  
 LANGUAGE: English  
 ENTRY DATE: Entered STN: 3 Dec 1999  
 Last Updated on STN: 5 Jun 2000

AB 1 Various classes of nitrovasodilators release nitric oxide (NO) through  
 distinct reaction pathways, many of which involve endogenous  
 reductants and/or oxidants. This study examined relaxations of  
 isolated rat coronary arteries induced by spermine NONOate (SPNO),  
 3-morpholinosydnonimine (SIN-1), nitroprusside (NP), S-nitroso-N-  
 acetylpenicillamine (SNAP) and nitroglycerin (NTG) in order to assess  
 whether their potency was influenced by any of six redox compounds: 1 mM  
 ascorbate, 1 mM dehydroascorbate, 0.1 mM dithiothreitol, 10 µM  
 diamide, 0.1 mM ferrocyanide, and 0.1 mM ferricyanide. 2 Only SPNO  
 spontaneously generated NO at measurable levels. These levels were  
 decreased by the presence of ascorbate and dithiothreitol, which  
 likewise decreased the potency of SPNO. 3 The potency of SIN-1 was  
 unaffected by any redox compound except ferricyanide, which increased the  
 potency not only of SIN-1, but also of other nitrovasodilators and  
 NO-independent vasodilators. 4 The potency of NP was decreased by two  
 structurally similar multivalent anions, ferrocyanide and ferricyanide,  
 suggesting that NP metabolism requires ionic binding to tissue. 5 SNAP  
 lost its potency in solutions containing ascorbate or dehydroascorbate.  
 SNAP potency was also decreased by the glutathione oxidant, diamide, and  
 by ferrocyanide and ferricyanide, suggesting that glutathione and ionic  
 binding may be required for NO release. 6 NTG appeared to relax arteries  
 via two pathways. One required only low concentrations of NTG and a  
 labile endogenous factor that was preserved by dithiothreitol  
 and eliminated by ferricyanide. A distinct second pathway required higher  
 concentrations of NTG. 7 These distinct attributes of nitrovasodilator  
 metabolism may underlie differences in regional specificity or tolerance  
 development, and therefore might eventually be exploited in the  
 development and use of nitrovasodilators.

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 concentrations of NTG. 7 These distinct attributes of nitrovasodilator  
 metabolism. . .

IT .  
 Circulation); Pharmacology  
 IT Parts, Structures, & Systems of Organisms

coronary artery: circulatory system

IT Chemicals & Biochemicals:  
ascorbate; dehydroascorbate; diamide; dithiothreitol;  
ferricyanide; ferrocyanide; nitric oxide; nitroglycerin;  
vasodilator-drug; nitroprusside; spermine NONOate: vasodilator-drug;  
SNAP [S-nitroso-N-acetylpenicillamine]: vasodilator-drug;  
3-morpholinosydnonimine: vasodilator-drug

RN 299-36-5 (ascorbate)  
10465-78-8 (diamide)  
3483-12-3 (dithiothreitol)  
13408-62-3 (ferricyanide)  
13408-63-4 (ferrocyanide)  
10102-43-9 (nitric oxide)  
55-63-0 (nitroglycerin)  
15078-28-1 (nitroprusside)  
136587-13-8 (spermine NONOate)  
33876-97-0 (3-morpholinosydnonimine)  
79032-48-7 (S-NITROSO-N-ACETYPENICILLAMINE)

ACCESSION NUMBER: 1993:323568 BIOSIS  
DOCUMENT NUMBER: PREV199396031918  
TITLE: Cardioprotective efficiency of dihydrolipoic acid  
in working rat hearts during hypoxia and reoxygenation:  
Phosphorus-31 nuclear magnetic resonance investigations.  
AUTHOR(S): Assadnazari, H.; Zimmer, G. [Reprint author]; Freisleben,  
H.-J.; Werk, W.; Leibfritz, D.  
CORPORATE SOURCE: Gustav-Embden-Zentrum der Biologischen Chemie, Klinikum der  
Johann Wolfgang Goethe-Univ., Theodor-Stern-Kai 6, W-6000  
Frankfurt/Main 70, Germany  
SOURCE: Arzneimittel-Forschung, (1993) Vol. 43, No. 4,  
pp. 425-432.  
CODEN: ARZNAD. ISSN: 0004-4172.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 12 Jul 1993  
Last Updated on STN: 13 Jul 1993

AB The working rat heart model was used for  $^{31}\text{P}$  nuclear magnetic resonance (NMR) studies during normoxia, hypoxia and reoxygenation. Aortic flows of about 35 ml/min could be achieved which equals 65% of the values obtained outside the NMR magnet. Addition of dihydrolipoic acid (DHL) at a concentration of 0.3  $\mu\text{mol/l}$  during hypoxia accelerated the recovery of aortic flow and stabilized it during reoxygenation. During hypoxia, inorganic phosphate contents (P-i) were significantly higher in controls. The phosphate shift indicated a pH decrease in control to 6.98, in DHL treated hearts the calculated pH was 7.15. During both hypoxia and reoxygenation, the phosphocreatine (PCr) contents were higher in the DHL treated hearts than in controls. In the controls, saturation transfer measurements revealed a decrease of the flux PCr  $\rightarrow$  ATP during initial reoxygenation, whereas after addition of 0.3  $\mu\text{mol/l}$  of DHL during hypoxia creatine kinase flux remained constant or increased. In isolated rat heart mitochondria, creatine kinase activities were measured under saturating and non-saturating concentrations of PCr. An increase in activity was observed under low PCr (non-saturating) conditions in the presence of 0.7 nmol DHL per mg of protein. At higher concentrations of DHL, creatine kinase activity was increased under all conditions. An increase in ATP synthesis in the working rat heart under influence of DHL is corroborated by NMR spectroscopy.